Case Report

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A case of brucellosis with severe thrombocytopenia

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ABSTRACT

Brucellosis is a zoonosis still endemic to a wide geographic area, including Turkey. It is characterized by multisystem involvement resulting in a variety of clinical signs and can also exhibit considerable variation in hematological terms. It can produce a variety of hematological manifestations, from pancytopenia to disseminated intravascular coagulation. Brucellosis must be considered, especially in endemic regions, in patients with symptoms suggesting infectious disease, undergoing investigation to identify the cause of thrombocytopenia. We present a case of brucellosis presenting with severe thrombocytopenia and recovering completely with treatment.

Keywords: Adolescent, brucellosis, thrombocytopenia

INTRODUCTION

Brucellosis is a zoonosis still endemic to a wide geographic area, including Turkey. It is characterized by multisystem involvement resulting in a variety of clinical signs and can also exhibit considerable variation in hematological terms. It can produce a variety of hematological manifestations, from pancytopenia to disseminated intravascular coagulation (DIC). Brucellosis must be considered, especially in endemic regions, in patients with symptoms suggesting infectious disease, undergoing investigation to identify the cause of thrombocytopenia.

We present a case of brucellosis presenting with severe thrombocytopenia and recovering completely with treatment.

CASE PRESENTATION

A 16-year-old female patient presented to our clinic due to acute onset of diffuse rashes all over her body, more pronounced on the arms and legs, over the previous 3-4 days and reported intermittent pain in the knees and legs, along with high fever and malaise exhibiting greater severity at night over the previous week. Anamnesis revealed that her family were farmers, and an animal pregnancy resulting in miscarriage had occurred. Physical examination revealed arterial blood pressure (brachial) 90/60 mmHg, heart rate 98 bpm (radial, rhythmic), and body temperature 37.4°C. We detected hepatomegaly and splenomegaly. Laboratory findings revealed thrombocytopenia with WBC 7100/mm³, Hb 12.3 g/dl, platelet 4000/mm³, and normal biochemical values. Peripheral blood smear and bone marrow aspiration revealed no findings suggestive of hematological malignancy. Bone marrow aspiration was normal except for megakaryocyte elevation. Peripheral blood smear was normal except for low platelet count. Tests for brucellosis were requested because of the joint pain, anamnesis and nocturnal fever. Infective endocarditis was excluded by echocardiography. Corticosteroid therapy was started with a diagnosis of idiopathic thrombocytopenic purpura (ITP). However, the platelet count did not improve during follow-up, and corticosteroid therapy was stopped. Rifampicin 600 mg/day and doxycycline 200 mg/day were started on the basis of positive Brucella coombs (1/640), Brucella tube agglutination (1/320) and Rose Bengal (1/320) tests. Additional tests for Brucella IgG and IgM performed were 45.31 and 34.68 (9 negative and >11 positive). This treatment resulted in the normalization of body temperature, and the platelet count rose to 202,000/mm³ within 2 weeks of follow-up. Appropriate treatment was prescribed, the patient's clinical signs improved and she was discharged. Oral rifampicin 300 mg tb 1x600 mg/day and doxycycline 100 mg tb 2x100 mg were used for 6 weeks. All her symptoms had resolved, and the following laboratory results were determined: WBC: 7700/mm³, Hb 12.3 g/dl, and platelets 267000/mm³. After the end of treatment, the patient was monitored in terms of risk of relapse for a 1-year period, and no problems were observed.

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DISCUSSION

Brucellosis remains an important cause of high fever in developing countries and in rural parts of developed countries. It is a multisystemic disease associated with a very wide range of symptoms (1). In a retrospective study of 78 cases of brucellosis by Ulug et al. (2) the major clinical symptoms observed were high fever, malaise, arthralgia, myalgia, and sweating, and the most common clinical signs were high fever (82%), hepatomegaly (28%), and splenomegaly (23%).

Brucellosis may also lead to a number of hematological complications including thrombocytopenia, pancytopenia, DIC, and leucopenia (3). Citak et al. (1) reported pancytopenia in nine out of 146 patients diagnosed with brucellosis, while only 5 had thrombocytopenia.

The most frequent hematological complications of brucellosis are leukopenia and anemia. Thrombocytopenia has been reported in 1% to 8% of cases (4).

Theoretically, the mechanisms causing thrombocytopenia accompanying any bacterial or protozoal infection include the direct platelet toxicity resulting from the platelet activating factors secreted by the micro-organisms, platelet damage due to endotoxins and exotoxins along with increased platelet clearance, adherence of the platelets to the damaged vascular surfaces, and immune-associated platelet destruction (5).

The exact mechanisms responsible for thrombocytopenia due to brucellosis are obscure. The proposed mechanisms include hypersplenism, intravascular coagulation, bone marrow depression due to septicemia, hemophagocytosis, formation of granulomas, and peripheral destruction (6). Hemophagocytosis and granuloma formation were not detected at bone marrow aspiration in our case. DIC was also not observed based on biochemical and hematological parameters.

Descriptions of brucellosis associated with severe thrombocytopenia in the literature take the form of case reports (7,8).

In a case report by Akinci et al. (7) a 23-year old female patient presenting with epistaxis, high fever, and severe thrombocytopenia was diagnosed after investigations. ITP was initially suspected in this case, and corticosteroid therapy was started, but the patient's clinical symptoms and laboratory findings failed to improve. Corticosteroid therapy was discontinued when Brucella abortus growth was observed in blood culture, and the patient was started on rifampicin and doxycycline. Platelet counts subsequently returned to normal. Immune destruction was considered as the mechanism responsible for thrombocytopenia. In that case we think that immune destruction may also have played a role in our patient. We further think that toxic effects on platelets through various inflammatory molecules released during the course of brucellosis may be involved as a cause of thrombocytopenia, in addition to immune destruction, in our case.

Similarly, a case of isolated thrombocytopenia was reported by Dal et al. (8). That patient was initially diagnosed with ITP and treated with steroids, but no improvement was observed. A diagnosis of brucellosis was subsequently established, accompanied by clinical and hematological improvement following appropriate antibiotic therapy.

CONCLUSION

The possibility of brucellosis should always be kept in mind during differential diagnosis of thrombocytopenia in patients living in or having visited areas endemic to this infectious disease.

ETHICAL DECLARATIONS

Informed Consent: Written informed consent was obtained from the patient(s) participating in this study.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Status: The authors declared that there was no conflict of interest in this study.

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